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ARCHIVES OF PEDIATRICS

A MONTHLY DEVOTED TO THE

DISEASES OF INFANTS AND CHILDREN

JOHN FITCH LANDON, M.D., Editor

LEADING ARTICLES IN THIS NUMBER

Lymphnode Tuberculosis
A Decisive Factor in Pulmonary Pathology.
(Continued from May Issue)

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The Prevention and Treatment of Seborrhea Capitis with an Antibacterial Emulsion containing Methylbenzethonium Chloride.

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LYMPHNODE TUBERCULOSIS A DECISIVE FACTOR IN PULMONARY PATHOLOGY

PH. SCHWARTZ, M.D.

Warren, Pa.

(Continued from May Issue)

3. *Further Observations Relative to the Discussion of Lymphonodogenous Bronchial Lesions.*

We mentioned that the findings and statements relating to the occurrence and importance of lymphonodogenous bronchial lesions of tuberculous origin, were received with hesitation and disagreement by certain authors. One of the controversial points was the *frequency of these lesions in western countries.*

The author listed lymphonodogenous bronchial lesions in 25 per cent to 30 per cent of his routine autopsy cases in Turkey. However, Könn (Freiburg-Br., Germany), stated in 1952 that in 500 routine autopsies, he was able to observe lymphonodogenous bronchial lesions in no more than seventeen instances, that is, in 3.7 per cent of the investigated cases. In 448 post-mortems performed on persons, 16 to 19 years of age, he detected eight cases only in which bronchogenous perforative bronchial scars of tuberculous origin were present, that is, in 2 per cent; Wurm (Wiesbaden, Germany) also stated in 1952 that perforative bronchial lesions occurred in only six cases, that is, in 0.8 per cent in his 800 routine autopsies—eighty instances of pulmonary tuberculosis included. Uehlinger (St. Gallen, Switzerland) in a paper (1942) describing seventy-two cases of late primary infections in adults,

reported on thirteen observations in which lymphonodogenous bronchial lesions occurred. Supported by these investigations, he estimated, after the appearance of my publications, that in Western Europe fresh tubercular pulmonary processes are accompanied in maximally 20 per cent of the instances by lymphonodogenous bronchial lesions.

To clarify these contradictions, the Department of Health of the German Federal Government in 1953 gave the writer and his associates Dhom and Wyss, the opportunity to investigate 191 routine autopsies in three general hospitals in Hamburg. We observed lymphonodogenous perforative bronchial lesions of tuberculous origin—mostly scars—in eighty-one instances. The scars were—in very old people—sometimes small, nevertheless, unmistakably pronounced in sixty cases. Scars indicating previous perforative processes were observed in all of the fourteen chronic pulmonary tuberculosis cases we encountered. Recent lymphonodogenous bronchial defects were revealed in eight instances in which death occurred following an acute relapse of a chronic pulmonary phthisis. The same frequency of lymphonodogenous bronchial lesions characterized 450 routine autopsies performed by the author in the United States during the past four years. Voegtli, (1954), employing the autopsy method devised by the author, investigated 300 routine autopsy cases at the Pathological Institute of the University of Vienna and observed in all ninety-two typical, perforative bronchial scars in sixty-five cases, that is, in 21.6 per cent of the examined instances. Brecklinghaus (Essen, Germany, 1955), was able to list fresh bronchial perforative lesions and perforative bronchial scars in forty-seven observations, out of a group of 124 autopsy cases, displaying old or recent tuberculous pulmonary changes. He revealed eleven instances of lymphonodogenous bronchial lesions, in a group of twenty-nine observations of primary tuberculosis.

Of great value, in this connection, are the following reports on clinical, bronchoscopial investigations. Hutchison and Graham (1949) recounted bronchoscopic investigations in twenty-eight cases of tuberculous primary infections in children. Obvious perforations were present in seventeen cases (60.7 per cent) and preliminary stages of the perforation in nine other observations. Lewenfisz and Margolisowa, in 1949, gave an account of endoscopic findings in fifty tuberculous children, 4 years to 16 years old. Perforative lesions were present in sixteen patients, that is, in 32 per cent of the cases while in twenty-five other instances

edema, infiltration, ulceration, and granulation processes of the bronchial mucosa were detected as manifestations of a lymphadenogenous lesion. Subsequent to streptomycin treatment, bronchial cicatrices were seen in seven children; however, in other cases, new perforative defects or fresh lymphadenogenous granulation foci appeared. Debbaudt, Carez, and Bruninx, in 1949, observed in systematic endoscopic investigations that lymph node perforations occur in 25 per cent of the cases during the primary and secondary periods of pulmonary tuberculosis. Carez and Bruninx, in 1950, reported on 138 children and young adults who were examined endoscopically. Fistulas were observed in thirty-three cases. The authors pointed out that even extended perforative defects may heal without leaving any rough traces. They stressed that the detection of fistulas is sometimes difficult because they are covered by a layer of mucus. The perforations may be observed shortly after the enlargement of the lymph nodes becomes manifest. Circumscribed pulmonary infiltrations in cases in which simultaneously large hilar shadows appear are characteristic for the presence of perforations. Kourilsky and his associates reported, in 1951, on their experiences with seventy-one tuberculous persons, 3 to 25 years old. (The authors believe that they dealt, in all these cases, with primary infections.) Radiologically visible parenchymal changes combined with tracheo-bronchial adenopathy were present in forty-seven patients. Lymph node perforations which the authors believed explained the appearance of parenchymal lesions could be endoscopically detected in thirteen cases. The authors emphasized that the number of cases in which they were able to observe fistulas directly, obviously does not correspond to the actual frequency of this lesion. They stated—contrary to Dufourt—that following lymph node perforations, manifold radiological pulmonary changes may be observed which start with an isolated focus and eventually extend to a massive consolidation, and that the pulmonary involvements observed in their patients resulted from perforative lesions of the bronchi in 57.4 per cent of the cases. Suter and Iselin, in 1951, reported having observed lymph node perforations in twelve cases in a group of 192 persons from 18 years to 51 years old who suffered from pulmonary tuberculosis. In nine other patients changes were present which probably also were caused by lymphadenogenous involvements. In one instance, the esophagus also was perforated. In another instance, an originally extensive perforative defect characterized by intense granulation healed within four weeks so well that only

an infiltrate with a smooth surface could be seen. "We have . . . observed healing processes which left almost no traces; these instances seem to be of considerable importance because they show that the detection of a perforative lesion depends . . . on the timing of the examination and that the right moment may be missed, which occurs more often than is generally supposed." The authors pointed out that a striking discrepancy may exist between radiological and bronchoscopical findings. "We have repeatedly observed that a perforation occurred just in the area in which the lymph nodes appeared harmless or the tomograph showed no changes and vice versa." They stressed that perforations may occur in bronchial sectors which are inaccessible to endoscopy. The authors observed small fistulas which remained open for a rather long time and thereby induced the development of massive bronchogenic dissemination.

Lévi-Valensi and his associates, in 1951, reported on twenty-four cases of endoscopically detected lymphadenogenous bronchial lesions in adults; eighteen patients were 18 years to 30 years old, the others between 30 years and 60 years of age. Various radiological pulmonary changes were present in twenty-two instances. The authors stressed that the site of the bronchial fistula corresponds to the location of the radiologically visible pulmonary changes and that this fact could be a promise that the pathogenesis of tertiary phthisis might one day be unveiled . . . .

Fischer, (1953), with his method of "aimed roentgen contrast filling," succeeded in showing typical parabronchial distentions, that is, lymph node cavities in twelve instances in a group of twenty-eight tuberculous patients who were affected by fresh, extensive, mostly segmental pulmonary infiltrations. In one case, a single lymph node cavity could be demonstrated although no other pulmonary changes were present. This investigator found, bronchographically, flat depressions and small funnel-shaped depots on the contours of the lobar and segmental bronchi in approximately 10 per cent of the cases in which, radiologically, calcified hilar lymph nodes could be detected; while no similar changes of the bronchial wall were observed in cases in which lymph node cicatrices of tuberculous origin were absent.

Jeune, Mounier and Béthénod (Lyon, France) in 1951 investigated fifty tubercular children displaying segmentary condensations. Bronchoscopic examination revealed lymphonodogenous bronchial lesions in all instances. Moczkowa and Pecherck (1955) bronchoscopically observed lymphonodogenous bronchial lesions in

427 cases out of 742 tubercular children. Lemoine (Paris, France, 1956) reported on a study of 622 tubercular children and adolescents. He observed perforative bronchial lesions in 30 per cent of these cases.

We wish to mention another important publication, that of Jondot (Lyon, France, 1953) who reported that he was able to find lymphadenogenous perforative cicatrices of the bronchial wall in ninety-seven instances (3 per cent) in a group of 2,800 persons, 20 years to 60 years old, submitted to a routine bronchoscopic examination.

If we consider the fact that only half the number of lymphonodogenous lesions of tuberculous origin observed in autopsies are located within bronchoscopical reach, we may assume that actually more bronchial foci than listed were present in the cases investigated by the authors mentioned above, and that, on the other hand, in many instances in which the endoscopic examination was negative or inconclusive, lymphonodogenous defects were present in pulmonary parts not accessible to bronchoscopic inspection.

The systematic employment of Fourestier's endoscope, combined with cinematographic visualization of the tracheobronchial and esophageal lumen, may mean the turning point in the detection of tubercular lymphonodogenous lesions. No doubt, the diminution of the technical difficulties in performing bronchoscopy, and a better knowledge of the anatomical aspects of lymphonodogenous tracheobronchial lesions of tuberculous origin will contribute to the progressively increasing accuracy of clinical examinations.

Bronchography (Lorey, Fischer) and planigraphy (Troisier and Bariéty, and Lowys) also are important methods in the detection of lymphonodogenous bronchial lesions. Surprisingly, however, simple routine radiographies display manifold changes caused by enlarged tuberculous lymphnodes which discharge into the bronchial system. (Del Rio).

Another point vigorously discussed during the past few years was the *pathogenic significance* of lymphonodogenous bronchial lesions in relation to pulmonary tuberculous condensations simultaneously present.

It has been stated that the present author observed about 100 cases of lymphonodogenous bronchial lesions of tuberculous origin before 1947, the year in which he began to understand the real significance of this phenomenon. Up to that time, he had been inclined to assume that lymphonodogenous bronchial lesions occur rarely and develop if massive tuberculous infections induce the appear-

ance of particularly extensive pulmonary involvements. This attitude was more or less characteristic of contemporary pathologists in general. Systematic comparative observations beginning in 1947 revealed, however, that the lymphonodogenous bronchial lesion always was the primary occurrence and that pulmonary parenchymal infiltrations developed subsequently. These results were then endorsed by hundreds of pathological anatomical observations in which all stages of the development of lymphonodogenous bronchial lesions, and of the subsequent segmental aspiration-infiltrations, could be followed step by step. Today the assumption that tuberculous pulmonary condensations extend in segmentary localization and that they are produced by aspiration infection following bronchial lesions is widely shared.

We mentioned clinical investigations performed by Carez and Bruninx (1950); by Jeune, Mounier-Kuhn and Béthénod; by Kourilsky; by Lévy-Valensi, as well as by Suter and Iselin in 1951, in which the pathogenetic connection between a previous lymphonodogenous bronchial lesion and a subsequent pulmonary segmentary involvement was observed bronchoscopically and radiologically. Other important clinical papers, published recently by Brügger (1955), by Soulas and Mounier-Kuhn as well as by Haefliger and Mark (1956), and by Tanner (1957) endorsed emphatically the views published by the author since 1948.

Pathological anatomical investigators, such as Uehlinger, Könn, Wurm, even if they felt unable to confirm the findings and opinions of the writer, reported on cases—mostly children—in which they explained the development of pulmonary condensations to be the consequence of lymphonodo-bronchogenous aspiration infections. Uehlinger seemed inclined to accept the explanation that tuberculous pulmonary involvements, in children and adults affected by primary tuberculosis, are mainly caused by lymphonodogenous bronchial lesions; he also emphasized the predominant pathogenic role of perforative bronchial lesions in old age tuberculosis. Nevertheless, he maintained that lymphonodogenous bronchial lesions are only of secondary ("episodic") importance in tubercular persons who are no longer children, yet have not passed the threshold of old age!

Brecklinghaus, too, accepted the pathogenetic connection between lymphonodogenous bronchial lesions of tuberculous origin and pulmonary changes in the course of a primary infection. This investigator observed in a group of ninety-five cases of post primary tuberculous processes in adults, fresh perforative bronch-

ial lesions in fourteen, and perforative bronchial scars in eighteen instances. Notwithstanding the fact that in almost every case in which Brecklinghaus detected bronchial lesions, segmentary pulmonary parenchymal changes were also present, he felt that a pathogenetic rapport could not be proved with certitude. We see that, while Brecklinghaus had no hesitancy in linking pulmonary involvements to lymphnodogenous bronchial lesions in cases of primary tuberculosis, he did hesitate to accept an analogous connection in the chronic pulmonary tuberculosis of adults.

Simon, the eminent authority in the field of childhood tuberculosis, concluded a comprehensive review in 1951 on "New Opinions on the Pathogenesis and Nature of Pulmonary Tuberculosis", with the following statements: "Clinical and radiological examinations revealed many examples confirming Schwartz' opinion on the significance of the lymphonodogenous perforative bronchial lesions in the origin and development of pulmonary tuberculosis . . . We see that different clinical processes, infiltrations, disseminations, caseous pneumonias, massive calcified foci, indurations, shrinking and bronchiectasis, eventually lead to a common pathogenetic denominator." Valuable endorsement of the writer's pathogenetic interpretations was given also by other pediatricians, such as Rietschel (1952), Erichson (1953), and Villar (1954). Critical views were expressed by Dufourt (1953, 1954, 1956).

Comprehensively reviewing the development of the discussion, we can state that *after a period of resistance and hesitation, the fact that lymphonodogenous bronchial lesions of tuberculous origin constitute a regular occurrence in both children and adults and that this phenomenon is responsible for many dominant features of pulmonary tuberculosis, slowly but surely is winning acceptance.*

We would like to conclude this chapter with a short review of the American literature on perforative lesions caused by tuberculous mediastinal lymphnodes. American authors: Green (1855), Barry (1885), Biggs (1890), Flexner (1893-94), Cone (1897), Northrupp (1898), Mathews (1900), Coakley (1910), Jackson (1910), Lockard (1913) are among the pioneers in this field. Later the following American observers published related papers: Hart (1923), Sante (1927), Schonwald (1928), Stoloff (1928), Bucher (1928, 1937), Myerson (1930, 1934, 1936, 1940, 1952) Stone (1931), McConkey (1934), Reichle and Frost (1934), Northrupp (1934), Eloesser (1934, 1935), Bilderback and Ashley (1935), Reinberg (1935), Ballon (1935), Oppenheimer (1935),

Faust (1936), Spivek (1936), Phelps (1936), Epstein and Ornstein (1936, 1938), Moorhead (1937), Stokes (1937), Sanes and Smith (1937), Samson and associates (1937), Cohen and Higgins (1937), Vinson and Toone (1937), Schneider (1938), Kernan (1938), McIndoe and associates (1939), Cohen and Wessler (1939), Terplan and Hyde (1940), Sharp and Gorham (1940), Jenks (1940), Jones and associates (1943, 1946), Meneghello and Smith (1943), Salkin and associates (1943), Moersch and Tinney (1944), Auerbach (1944, 1949), Pray (1944), Meissner (1945), Silverman (1945), Holinger and Ringby (1946), Schwartz (1948-57), Aufses and Neuhof (1948), Clerf (1950), Coleman and Bunch (1950), Halle and Blitz (1950), Jenkinson and Bate (1951), Overholt, Wilson and Schwartz (1952), Hardy and associates (1952), Daly and associates (1952), Rubin and Harris (1955).

In 1934 Scobie published a comprehensive paper which even today, because of its excellent historic review, is indispensable. Rich was one of the first, in 1934 and again in 1946 to stress the occurrence and pathogenetic role of lymphonodogenous bronchial lesions. There are, however, only a few publications as yet in this country directly contributing to the new discussion.

Overholt and Wilson, in 1952, emphasized the importance of the present author's observations in regard to therapy.

Laft and associates published, in 1951 and in 1955, the results of investigations performed in eighty children. Among fifty cases showing clinical and radiological signs suggestive of pulmonary tuberculosis, positive bronchoscopic findings could be demonstrated in forty-one; among the latter, evidence of perforation was demonstrated in 46 per cent. The authors conclude that bronchial changes resulting from mediastinal lymphnode involvement constitute an important and increasingly recognized factor in the pathogenesis of primary pulmonary tuberculosis in children.

High and Nelson, in 1955, reported on observations in sixty-seven tuberculous infants and children examined bronchoscopically. Inflammatory changes in the tracheal and bronchial mucosa as well as extra luminal compression with inflammatory changes in the mucosa were observed most frequently. Endobronchial granulations were encountered in fourteen instances. Erosions of the bronchial wall, secondary to inflammatory changes in adherent lymphnodes, were not observed in this series.

Lincoln and associates (1955) observed endobronchial lesions bronchoscopically in 147 patients. External compression was

present in twenty-nine instances, narrowing of lumen in thirty-three, granulation tissure in eighty-six, ulceration in eighteen, caseous material in forty-three, erosion of bronchial wall in twelve cases. Eighty-four per cent of the group with endobronchial disease were less than 4 years of age.

Terplan, in 1955, at the same Symposium on Tuberculosis in Infancy and Childhood (National Jewish Hospital, Denver) at which Lincoln and associates, as well as Laff and associates reported on the findings mentioned above, stressed that he has observed during his long activity as a pathologist in Buffalo no more than four cases of lymphonodogenous bronchial lesions of tuberculous origin. In his opinion, the rupture of a caseated lymph node into a bronchus has very little, if any, significance. Also, he disclosed that he was able to demonstrate, occasionally, scars in the wall of the major bronchi, obviously caused by perforating lymph nodes, "largely as incidental findings without any significant tuberculous involvement of the lungs."

COMMENT

The new facts and the new discussion related to the occurrence and pathogenic importance of lymphonodogenous bronchial lesions and of lymphonodo-broncho-pulmonary changes, progressively transformed our thinking on pulmonary tuberculosis. Questions on the origin and pathogenic importance of apical tuberculosis or of subapical tubercular foci, which were intensely discussed for decades without being definitely solved, lost their predominance, as we were able to show that both apical and subapical tuberculous infiltrates develop following lymphonodogenous bronchial lesions. Also, these observations reduce the meaning of a discussion on the hematogenous origin of initial foci of chronic tuberculosis in adults. We found that the relapse in the case of a chronic tubercular pulmonary affection is, in general, induced by the bronchial discharge of necrotic lymphnodes in which old tuberculous foci exacerbated, or which were involved by the extension of the tubercular inflammation, originating from other lymphnodes, or from old, reactivated pulmonary foci.

The new observations resulted in the definition of many changes hitherto unknown, or insufficiently appreciated: lymphonodogenous tracheobronchial penetration foci, perforative ulcers and scars of the tracheobronchial system, segmental aspiration-infiltrations of the pulmonary parenchyma, etc. No doubt, the endeavor to recognize all these attributes of pulmonary tuberculosis will

enliven and enrich the clinical pathology. Surprisingly often we observed enlarged parapulmonary lymphnodes and lymphnode cavities in clinical radiographies of cases displaying these changes in autopsies.

Another point which we would like to emphasize here is the essential similarity between childhood primary tuberculosis and the initial changes in adults subjected to a relapse of a chronic pulmonary process. We learned that pulmonary foci, resembling typical primary affects in children, may develop in adults subsequent to perforative bronchial lesions induced by exacerbated tubercular lymphnodes. Another important new problem is the bilateralization of tuberculous pulmonary involvements. We observed that in cases in which tuberculous pulmonary disease begins on the left side, it reaches—by the intermediary of parapulmonary lymphnodes—in a short lapse of time the right lung, causing lymphonodo-bronchogenous pulmonary involvement. On the other hand, pulmonary tuberculosis beginning on the right side displays a marked tendency to remain restricted to this lung. These observations suggest that while the lymph circulation extends easily from the left side to the right, it is blocked in its flow from right to left.

We realized that a paratracheal lymphnode complex, situated on the right side below the subclavian artery, as well as the bifurcation lymphnodes, constitute centers of decisive importance in the pulmonary lymph circulation. These groups often contain active tubercular foci in cases in which the process healed in all other localizations. On the other hand, these same lymphnodes are in many instances the origin of exacerbative processes. In cases in which these two lymphnode complexes completely disappeared following their discharge into the tracheobronchial system, we repeatedly observed the spontaneous healing of very extensive pulmonary tuberculosis (Fig. 6). These types of pathological-anatomical experiences certainly endorse surgical endeavors (Giraud, Métras and associates (1953, 1956) to abbreviate and heal tubercular pulmonary involvements, by elimination of parapulmonary lymphnodes.

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FLUID REGULATION IN NEPHRITIS AND NEPHROSIS OF CHILDHOOD. (Minnesota Med., 37:269-274, April 1954). The author considers the treatment of children with acute nephritis, chronic nephritis, and the nephrotic syndrome. Using the concepts of fluid and electrolyte metabolism contributed by Gamble, he establishes a rational course of administration of salt and water. Only in anuric or oliguric states associated with renal disease should intravenous administration of fluids and salt be rigidly restricted. Fluids, when ingested orally, seldom produce aggravation of oliguric or anuric states. In chronic nephritis, if edema is absent, the intake of salt and water need not be restricted. Slight hope is offered for increased recovery rates among patients with the nephrotic syndrome through judicious use of corticotropin and cortisone. Additional help toward improvement of recovery rates can be expected from antibiotics. Complete exclusion of salt from the diet alone can produce edema and oliguria. Treatment of the low salt syndrome requires administration of hypertonic salt solutions.—*J.A.M.A.*

THE PREVENTION AND TREATMENT OF SEBORRHEA CAPITIS WITH AN ANTIBACTERIAL EMULSION CONTAINING METHYLBENZETHONIUM CHLORIDE*

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and
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Seborrhea capitis (cradle cap) is one of the common dermatologic diseases of infancy and early childhood. It is characterized by the presence of greasy scales and crusts on the scalp. In mild cases there is a slight scaling present over a small area about the anterior fontanelle. In severe cases the entire scalp may be covered with a thick-matted layer of scales and crust which are difficult to remove with ordinary soap and water shampoo. When scraped off with a fine comb the underlying skin is found to be erythematous and may bleed. When this rash extends down onto the face and body it is termed seborrheic dermatitis or seborrheic eczema. It is seldom seen before four weeks of age and practically never in the newborn infant. If untreated, seborrheic capitis may last for several years. Secondary infection is a common complication.

The etiology of seborrhea capitis has never been clearly established. It was once believed to be due entirely to uncleanliness and neglect. However, "cradle cap" can often be seen in infants who have the best hygienic care. A common opinion is that it is primarily an allergic dermatitis. When it is prominent on the face it may be almost indistinguishable from atopic eczema in appearance. However, it is not pruritic and responds rapidly to local therapy and not to an elimination diet. It is doubtful that seborrheic dermatitis is an infection caused by a specific organism since no causative bacteria have ever been demonstrated. Since numerous fat glands are concentrated in the scalp, many observers have felt that there may be some dysfunction and overactivity of fat secretion in this area. Hereditary, endocrine and hormonal factors have been considered in the etiology but no definite information is actually available on this point.

At present, therapy consists in the use of keratolytic agents applied locally in an ointment base. The drugs commonly used are

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salicylic acid, benzoic acid, resorcinal, sulfur and tar. Selenium suspensions have also been advocated. Selenium is a potentially toxic drug and its use must be restricted to once a week. At times the other drugs used may cause local irritation, consequently, the tendency has been to use these drugs sparingly and limit them to use in more severe cases. The use of simple lubricants such as petrolatum, mineral oil and olive oil has not been effective. Prophylaxis of seborrhea capitis has been neglected because the use of keratolytic drugs for long periods of time on sensitive infants' skin is not desirable and the use of lubricants and shampoo has not prevented "cradle cap" successfully.

Seborrhea capitis is quite common and, therefore, is seen frequently in our clinics. Because of its frequency and since prevailing therapy is not completely satisfactory, we decided to study the efficacy of an antibacterial emulsion containing methylbenzethonium chloride in an oxycholesterin absorption base of liquid petrolatum and lanolin (Cradol®)†. This product is designed to soften and separate scales and crusts from the scalp so that they may be easily removed by washing and brushing. The methylbenzethonium chloride tends to prevent infection and permits greater penetration of the softening emulsion. It appears to be nontoxic, hypoallergenic and nonirritating.

Method of Study: Sixty cases of seborrhea capitis of varying degrees of severity were studied. Many of these had already been treated with petrolatum. Thirty were treated with the antibacterial emulsion (Cradol®) labelled "A" and thirty were treated with a lotion containing salicylic acid labelled "B". The cases were alternated. A control card was made for each child showing the name of the patient, age and sex; the treatment followed, i.e., whether Preparation "A", Preparation "B" or Petrolatum; area of the "cradle cap", whether mild, moderate or severe, and the duration and response to treatment; whether any recurrence; reactions and comments. Wherever possible cases of equal severity were placed in each group. Infants varied in age from 3 weeks to 15 months. There was one child of 8½ years who had a dry scalp. Treatment averaged about 10 days and patients were kept under close observation by the physicians.

Patients in both groups were instructed to apply medication by gentle massage into the scalp twice daily for three days, followed with a shampoo using a mild soap and then fine combing the scalp. This procedure was repeated several times if necessary.

†Cradol is the product of the Homemakers' Products Corporation, New York, N. Y.

Patients were classified as cured when the scalp appeared entirely clear of all scales and crusts, and the skin was normal in color and texture.

Patients were classified as improved if the amount of scales and crusts were greatly reduced, but not completely cured.

Those patients with crusts and scales as extensive as when treatment was begun were classified as not improved.

Results:

	Cases Cured	*Improved*	*No Improvement*	*Av. days of Rx necessary*	*Recurrence*	*Reaction*
CRADOL (Prep. "A") Total 36 cases	26	8	2	5	4	0
Lotion containing salicylic acid (Prep. "B") Total 30 cases	12	10	8	10	11	2

Of the 8 resistant cases which did not respond to Preparation "B", six were treated with Preparation "A", (Cradol®), of which four were cured and two failed to respond to treatment.

There were no reactions with Preparation "A" (Cradol®); there was no evidence of any irritation, toxicity or allergenicity. Two infants who failed to respond to treatment with Preparation "B" developed moderate erythema of the scalp after the second course of treatment.

COMMENT

Although generalized seborrheic dermatitis is relatively infrequent, seborrhea capitis (cradle cap) in infancy and children is quite common. In the past, the treatment and prevention of seborrheic dermatitis have been somewhat retarded by the fact that keratolytic drugs have been used rather sparingly because of the tendency to local irritation. However, we believe that all cases of seborrhea capitis should be treated both for esthetic reasons and also to prevent the possibility of secondary infection. It is also quite possible that treating "cradle cap" early may prevent more serious and extensive seborrheic dermatitis.

In general, cleansing of the scalp in newborn infants is some-

what overlooked in routine skin care because many mothers fear injury to the anterior fontanelle. The routine application of an emulsion containing methylbenzethonium chloride in an oxycholesterin absorption base of liquid petrolatum and lanolin (Cradol®) as part of the daily cleansing will greatly reduce the incidence of seborrheic dermatitis.

SUMMARY

1. A new emulsion for prevention and treatment of seborrhea capitis (cradle cap) is described.

2. Sixty infants and children with "cradle cap", ranging in age from 3 weeks to 15 months, were studied.

3. Of thirty-six infants treated with the above product, twenty-six were cured and six improved.

4. The product is nontoxic, hypoallergenic and nonirritating in the prophylaxis and treatment of seborrhea capitis.

INFECTIOUS HEPATITIS IN CHILDREN. C. G. Grulee and H. P. Brawner. J. Louisiana M. Soc., 107:188-193, May 1955. Infectious hepatitis is a disease of prime importance to any physician who cares for children. Its greatest incidence is in children between the ages of 4 and 14 years. The severity of the disease is as a rule directly proportional to the age, ranging from mild cases in infants without jaundice to severe cases in older jaundiced children. The vague symptoms of the nonicteric form frequently occurring in infants and young children are particularly difficult to recognize. Even though the clinical course of the disease in these young persons is often prolonged, they seldom suffer permanent liver damage. The fact that they excrete virus in their stools over many months constitutes a difficult problem in preventive medicine which has not yet been solved. The treatment of infectious hepatitis in children, as in adults, is almost entirely symptomatic. Prevention of the disease is possible by careful attention to personal hygiene and by the use of gamma globulin. Effective levels of passive immunity are obtained with doses of gamma globulin as small as 0.01 cc. per pound of body weight if given at least seven days before the expected onset of jaundice. Protection can be expected to persist for as long as six weeks.—*J.A.M.A.*

CLINICAL REVIEW

In order to encourage the writing of clinical articles by recent graduates or senior medical students, the ARCHIVES *will publish monthly at least one such paper from the classes of Doctor Lawrence B. Slobody, at New York Medical College, New York, and Doctor Walsh McDermott, at Cornell Medical School, New York. Other interested medical schools are cordially invited to submit student papers for consideration.*

CONGENITAL ATRESIA OF THE ESOPHAGUS WITH TRACHEO-ESOPHAGEAL FISTULA. A REVIEW.*

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and
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INTRODUCTION

While innumerable articles are found in the literature dealing with the esophageal atresias, with and without associated tracheo-esophageal fistula, a comprehensive survey of the subject is timely. Accordingly, this presentation has been prepared with emphasis on diagnosis, treatment, and prognosis.

HISTORY

Simple atresia of the esophagus was first recorded by Durston[1] in 1670. Thomas Gibson[2] observed a baby with congenital atresia of the esophagus with tracheo-esophageal fistula in 1696 and examined it at postmortem. He did not publish the record of the case until 1703 when he described it in detail and noted the feeding difficulty. In 1880, Mackenzie[3] found forty-two cases in the literature, and in 1919, Plass[4] found one hundred and forty-nine. The increased frequency was probably because of increased diagnostic acumen. Gross[5] considers esophageal atresia a relatively common anomaly.

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This paper received the first award for fourth year medical students' presentation of a pediatric problem.

CLASSIFICATION

Although there are many ways of classifying this anomaly, the most widely used classification is that of Vogt[6] schematically represented in diagram I. Type I: Complete absence of the esophagus. Type II: Atresia of the esophagus with an upper and lower esophageal segment each ending in a blind pouch. Type III: Atresia of the esophagus with tracheo-esophageal (a) with the fistula between the upper esophageal segment and the trachea; (b) with the fistula between the lower esophageal segment and the trachea; (c) with a fistula between both esophageal segments and the trachea.

To the above classification may be added the very uncommon tracheo-esophageal fistula without atresia of the esophagus.

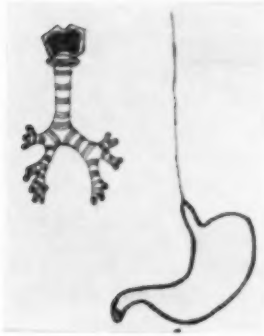
Of the various types described, Vogt's Type III-B is the most common with only occasional cases varying from this form. Gross[5] and Leven[7] in their respective series reported an incidence of more than 90 per cent of this type.

EMBRYOLOGY

The esophagus is developed from the first part of the primitive gut, just caudal to the pharynx. In the 3mm embryo, a median ventral groove develops in the floor of the pharynx at the level of the most posterior pair of pharyngeal pouches. This "laryngotracheal groove" is rapidly transformed into a tubular mass of entoderm parallel to the digestive tract and is prolonged caudad, eventually giving rise to the larynx, trachea, and lung buds. By the 4mm stage, the pulmonary anlage has grown caudally so that two lateral longitudinal furrows are formed in the wall of the foregut. Gradually these furrows, acting as a septum, begin to separate the foregut into an anterior trachea and a posterior esophagus. The respiratory primordium is completely separated from the esophagus by the 11mm stage.[8, 9]

Why the separation of the trachea from the foregut should in some cases develop into atresia, with or without fistula, has been explained on the basis of many theories. Each depends on a failure of a critical step in the embryologic development of the esophagotracheal anlage. No theory is fully satisfactory.

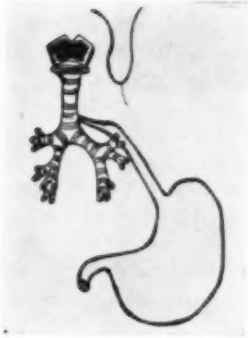
One theory is based on a peculiar growth phase in the early development of the intestines, particularly the esophagus. At about the 20mm stage, there is a tendency for the epithelial lining of the primitive gut to grow exuberantly before the size of the primitive



TYPE I.

TYPE II.

TYPE III-A.



TYPE III-B.

TYPE III-C.

Fig. 1. Schematic representation of Vogt's classification.

lumen can accommodate it. Thus, masses of epithelial cells tend to obliterate the lumen forming a solid bar of tissue. Normally, this stage is followed closely by enlargement of the lumen of the gut, with a spreading out of the epithelial cells. Cessation of growth at this state of transitory closure may be causally involved in instances of congenital atresia. Singleton and Knight[10] feel that this theory is untenable because the "solid bar" stage does not appear until the 20mm embryo, after esophago-tracheal anlage is well formed (4-6mm stage).

A second theory is based on mechanical deviation of the lateral furrows or septa by pressure from an enlarged heart or anomalous vessels. Thus, the lateral septa would be deviated posteriorly to fuse with the dorsal wall of the foregut and result in atresia of the esophagus.[10]

Rosenthal[11] believes that the explanation for this anomaly lies in an altered cell growth along the lateral septa and not mechanical deviation. With deficient or absent growth of the septa there would be development of a fistula, while failure of growth of entodermal cells of the dorsal wall of the foregut would result in esophageal atresia.

Finally, there is a third theory by P. Gruenwald,[12] who believes that the almost constant association of atresia and fistula is the outstanding feature of this malformation (Fig. 2). Any theory explaining its genesis must account for this combination of atresia and fistula. This anomaly is a mere disturbance of the coordination of normal developmental processes. After the respiratory primordium has reached the diverticulum stage, two things take place. First, the respiratory anlage is separated from the foregut. This proceeds in a caudo-cranial direction, leaving only the proximal portion of the original communication to form the "aditus ad laryngum". Then a second process takes place. This is a rapid growth in a cephalo-caudal direction of the laryngotracheal tube. At this time the laryngotracheal tube is growing at a faster rate than the esophagus. With this difference in growth intensity, normal development depends on the early separation of both primordia. Should the esophagus remain attached to this rapidly growing laryngotracheal tube, a portion of the esophagus will be drawn out into a narrow strip of tissue corresponding to and contributing to the formation of the dorsal wall of the trachea.

The upper atretic part of the esophagus would develop from that portion of the primordium which remains attached to the

pharynx. The normal caudal esophagus would originate from the caudal end of the composite tube. Thus we have formed an atresia of the esophagus with an associated tracheo-esophageal fistula. There has been much research lending support to Gruenwald's theory, such as the finding by Gutman[13] and others of esophageal structures in the dorsal wall of the trachea.

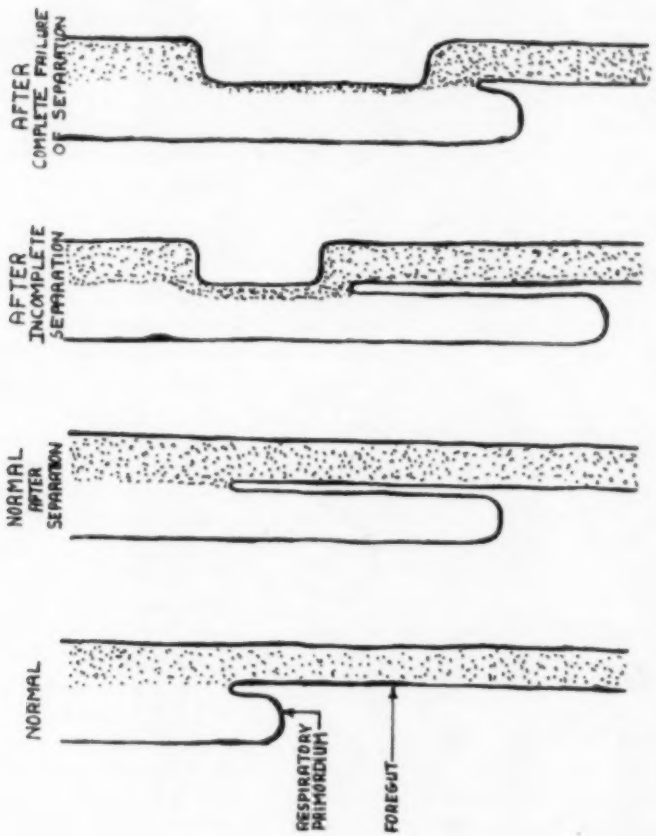


Fig. 2. After Gruenwald (12).

Failure in the growth process may well be a genetic factor in tracheo-esophageal fistula as is shown in the case reported by

Mackenzie,[3] where a man fathered three infants, all with the anomaly by different wives. Lanman[14] reported a case in a brother and sister, and Grieve and McDermott[15] described the anomaly in each of two brothers.

SIGNS AND SYMPTOMS

The earliest and most common sign of esophageal atresia is excessive salivation. The saliva exudes from the mouth because of the blocked swallowing mechanism.[5] Mucus accumulates in the pharynx resulting in respiratory obstruction.[16] Frequent suctioning is required. When fed, the infant hungrily takes food, but swallowing is immediately followed by gagging, coughing and vomiting. There is occasional temporary cyanosis and dyspnea, especially on feeding. Whenever excess salivation or dyspnea is seen in the newborn, the patency of the esophagus should be investigated.[5] Abdominal distention may or may not be present.

DIAGNOSIS

Delay in diagnosis results in deterioration of the condition of the patient, delayed treatment and increased mortality. Early diagnosis is stressed. The diagnosis should be suspected from the above signs. Investigation should be started at once by the passage of a small urethral catheter, the passage of which is stopped by the obstruction. Confirmation of the diagnosis is by roentgenography. The tracheo-esophageal fistula may show on lateral exposure as a pencil-like airway. A hypertrophied esophageal pouch may displace and narrow the trachea anteriorly. Air absence in the proximal esophagus resulting from secretion accumulation is occasionally seen. Further help is gained by demonstration of catheter passage obstruction.

Contrast media provides the most satisfactory means of visualizing the defects. The use of barium for visualization should be avoided. It produces severe irritation of the lungs which may result in death. Air instilled by means of the catheter is of help. The use of bland iodized oil has been found to give the best visualization. The latter procedure is, however, not without danger for such oil, especially in large amounts in abnormal lungs, may initiate pneumonic changes. Only about one milliliter or less of oil should be used. After administration, the patient is kept prone. This reduces the chance of aspirating the oil. Immediately after the roentgenographic examination, the oil should be removed by suction using fluoroscopy as a guide.

The initial examination is by fluoroscopy. Films are then taken in both the antero-posterior and lateral views for the detection of possible fistulas and demonstration of esophageal atresia (Figs. 3

Fig. 3. Anteroposterior view demonstrating atretic esophageal pouch with lipiodol.

Fig. 4. Lateral view. Pouch again demonstrated with lipiodol.

and 4). Pulmonary condition is also noted. Atelectasis may be found and is of two varieties:

1. Fetal, which for the most part is in the right upper lobe;
2. That secondary to aspiration.

Pneumonic processes in the lungs may be difficult to discern for about one-half of the babies with tracheo-esophageal fistulas have increased densities in the lungs.

The roentgenographic appearance of the stomach and small bowel is variable. Air in the stomach and intestines with complete congenital atresia denotes a tracheo-esophageal fistula (Fig. 5).

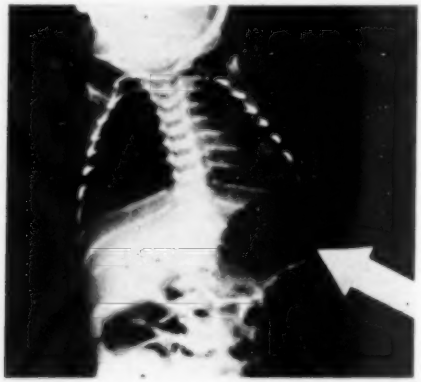


Fig. 5. Flat plate showing stomach dilated with air.

However, absence of air does not necessarily mean that a fistula is not present. Undue distention is not a prominent sign, although in some cases it may result in respiratory distress. It should be remembered that air in the gastrointestinal tract is physiologic at birth.

Purposeful instillation of a dye by means of intratracheal catheter to outline tracheo-esophageal fistulas is contraindicated.[16]

DIFFERENTIAL DIAGNOSIS

Prominent in the symptomatology of esophageal atresia is vomiting. If the onset of this vomiting is early in life and especially if there is distention with failure to pass meconium, several anomalies of the gastrointestinal tract should be considered.

(a). Tracheo-esophageal fistula without atresia which results in repeated episodes of respiratory embarrassment associated with feeding. Air from the fistula produces gaseous distention and there is pulmonary distress from regurgitation through the fis-

tulous tract. The fistula may be closed part of the time causing intermittent signs and symptoms. Radiologic procedures establish the true nature of the condition.

(b). Achalasia, spasm of the cardia, interferes with the passage of food into the stomach and regurgitation occurs.

(c). In chalasia or relaxation of the cardia, regurgitation is found only when the patient is lying flat. The latter two entities may be differentiated by an esophagogram.

(d). Esophageal diverticula are extremely rare and are apparent on roentgenographic studies.

(e). Diaphragmatic hernia may result in rapid respirations and cyanosis from pulmonary compression. The chest moves less freely on inspiration and is often dull to percussion. The heart may be pushed to the opposite side and bowel sounds may be heard. Scaphoid abdomen is noted. Ordinary chest plate establishes the diagnosis.

(f). Duplication of the stomach is another rare cause of vomiting.

(g). Duodenal atresia or stenosis produces signs of high intestinal obstruction with abdominal distention. The baby vomits within twenty-four hours following delivery, but the vomitus contains bile. On roentgenographic examination a characteristic "double bubble" is seen. Malrotation of the colon, annular pancreas, atresia or stenosis of other parts of the small bowel and meconium ileus (mucovescidosis) may also result in small intestinal obstruction.

(h). Pyloric stenosis, pylorospasm and adrenocortical insufficiency likewise cause vomiting, but this is usually after the first week of life.[17]

TREATMENT

I. *Surgery:* Early therapeutic attempts with gastrostomy or jejunal intubation for feeding failed. Closure of the distal esophagus to prevent regurgitation by either stenosis or ligation, combined with gastrostomy, was unsuccessful.[8]

In 1939 the survival of the first case of tracheo-esophageal fistula occurred. Leven and Ladd,[18] working independently, developed a multi-stage procedure to deal with the problems of esophageal atresia with fistula. Temporary diversion of the sa-

livary stream, closure of the fistula and gastrostomy for feeding were done at one setting. A plastic esophagus was then constructed at a later date. This plastic esophagus was at first antethoracic, then placed in the anterior and finally the posterior mediastinum. The latter, as advocated by Swenson,[19] is now done in all multi-stage constructions.

Primary anastomosis is the ideal solution to the problems caused by esophageal atresia with fistula. This procedure lessens hospital stay and complicated surgical and nursing care. Although attempted by Lanman[14] in 1936-1937, the first successful anastomosis of this type was accomplished by Haight[20] in 1941. Primary anastomosis is used now by some surgeons in all cases, if necessary, by bringing up the proximal stomach.[5] However, others hold that the latter procedure carries with it too high a mortality rate to warrant its use. In such cases the multi-stage procedure is used.[7]

II. *Preoperative care:* In the past when the diagnosis of esophageal atresia and tracheo-esophageal fistula was made it was considered a surgical emergency. Today, however, it is generally agreed that it is unwise to operate immediately. These infants are often in poor physical condition. Many have come considerable distances to reach the surgeon and are starved or have pneumonia. The babies are poor surgical risks. It becomes imperative to get them into better physical condition, thus improving the chance of successful surgical results. Although the optimal time for surgery is within the first 24 hours of life, Gross[5] and others advocate deferring operation for 18 hours to 36 hours if necessary in order to give the infant the adequate preoperative care.

In the preoperative management of these infants the following points are of importance.

(a). *Positioning the patient:* Gross[5] and his group advise the semi-sitting position. Since in the most common from of this anomaly there is a fistulous connection between the lower esophagus and trachea, this position would reduce the tendency of gastric juices to regurgitate into the tracheo-bronchial tree and thereby lessen the chance of chemical pneumonitis. On the other hand, Leven, et al.[7] favor the prone position with the head dependent. This increases drainage of salivary secretions and reduces the chances of aspiration pneumonia.

(b). *Continuous or frequent suctioning of the blind proximal esophageal pouch:* A No. 8 urethral catheter is used. It appears

that the semi-sitting position suggested by Gross in combination with adequate suctioning of the proximal esophagus is probably the best plan.

(c). *Alleviation of respiratory distress:* The infant should be placed in an oxygen tent. Care must be taken not to apply positive pressure since this might lead to further embarrassment of respiration due to inflation of the stomach through the fistula.

(d). *Intravenous therapy:* A cut-down over a vein should be performed and a polyethylene tube secured in the vein. Parenteral fluids should be administered in order to meet fluid and electrolyte requirements. However, the total amount of fluids, and particularly sodium, should be strictly limited. This will avoid the formation of pulmonary or generalized edema which often occurs in these infants when an excess of fluid is given. Leven[7] suggests that 75 ml. of fluid per kilogram per 24 hours be given. One-third of this amount is given as normal saline or Darrow's solution, and the remainder as five per cent glucose in distilled water. Gross[8] recommends ten per cent glucose in distilled water to meet caloric requirements and bases his total fluid requirement at 10 ml. per pound per 12 hours.

(e). *Blood transfusion:* If the hemoglobin level is less than 15 grams, whole blood is indicated. On the other hand, if the hemoglobin lebel is greater than 15 grams, plasma may be used instead. It should be kept in mind, however, that both whole blood and plasma are sodium-containing solutions and the amount of each given must be accounted for in calculating the total sodium requirement.

(f). *Antibiotics:* Intramuscular combined antibiotics should be given. Gross[5] advises procaine penicillin 20,000 units every eight hours and streptomycin ten milligrams per pound.

(g). *Vitamins:* It has been recommended that vitamin C and vitamin K in therapeutic doses be given to the infant.

III. *Anesthesia:* As in any major surgical procedure, good anesthesia is one of the prerequisites for the successful outcome in cases of esophageal atresia. Anesthetic problems revolve about adequate oxygenation and control of lung inflation, efficient elimination of carbon dioxide, adequate exposure with quiet operative field, maintenance of adequate circulating blood volume by prompt replacement of blood loss, control of secretions and bleeding into the tracheobronchial tree, and finally, rapid emergence from anesthesia without depression of respiration.

In the immediate preoperative period no medications are given for these contribute to complications. At the time of surgery, a plastic catheter should be in place in a leg vein. Prior to the induction of anestesia, a tracheobronchial toilet, aspiration of the main bronchi and trachea is performed.

For induction of anesthesia, endotracheal intubation without anesthesia is preferred. This method permits adequate ventilation with an open chest. It minimizes dead space and permits prompt aspiration of tracheal secretions. Furthermore, anesthesia can be maintained in a light plane, not disturbed by laryngospasm. Induction in this manner is shorter because breath-holding with resultant cyanosis is avoided. Although some[5] feel intratracheal tubes should be avoided for they are apt to set up postoperative irritation in the larynx and trachea and may permit development of high pulmonary pressures which cause overdistention of the alveoli resulting in pulmonary edema; others report no respiratory obstruction from laryngeal edema.

Intubation should be as atraumatic as is possible. A non-rebreathing valve is connected to the endotracheal tube. This minimizes dead space and allows efficient carbon dioxide elimination. Moreover, there is absence of heat retention or production. Respirations may be efficiently controlled or assisted.

It is universally agreed that cyclopropane is the anesthetic agent of choice. It is not irritating, induces quiet respiration, is easily controlled and has little effect on circulation and metabolism. Emergence from anesthesia is rapid. Anesthesia is begun with cyclopropane-oxygen mixture. This may be followed by a switch to ether bubbled through a vaporizer.

Blood replacement is a difficult problem, for under and over replacements are equally dangerous. Seventy milliliters of blood in a two-kilogram infant are equivalent to 1000 milliliters in an adult of 70 kilograms. Thus, operative loss is replaced milliliter for milliliter. Weighing of sponges may be helpful. Most of the blood losses occur in the opening of the chest wall, where from ten to forty milliliters are lost. The blood loss ranges from ten to two hundred and fifty milliliters with an average of eighty-five milliliters.

Anesthesia is maintained as light as possible with the concentration of cyclopropane needed varying with each individual. Infants up to twelve hours of age need almost no anesthesia. Respiratory assistance is given from the beginning of the operation

to decrease effort and to avoid mediastinal shift once the thorax is opened. Because of circulatory effects, d-tubocurarine is not recommended. At the time of anastomosis, anesthesia should be deepest to prevent swallowing which draws the upper esophageal segment up and may result in the sutures cutting through. At the conclusion of the operation, inflation of the atelectatic lungs, especially on the right, is time well spent. Careful extubation is preceded by tracheal aspiration and adminstration of 100 per cent oxygen. The wound dressing should be small to allow the chest to expand freely.

Anesthetic complications may result from the tube being passed too deeply so that it gets into the bronchus and materially diminishes the available respiratory surface. Tube occlusion, by as little as one or two drops of mucus, is a constant threat and meticulous aspiration must be carried out throughout the entire operation. Because the tubes may become obstructed despite aspiration, emergency replacement tubes should always be handy. In the course of surgery one of the bronchi may become kinked. When respiration suddenly becomes difficult, this must be thought of. Oxygenation of the blood may be evaluated by its color. With no respiratory difficulty, presistent dark blood suggests congenital malformation of the heart.

Circulatory complications may be difficult to recognize. For example, incipient shock in infants is rarely accompanied by an increase in pulse rate; more often, pulse and blood pressure suddenly become unobtainable. The heart rate slows decidedly. Treatment is best effected by increased ventilation of the lungs with 100 per cent oxygen and blood under pressure. Circulation must not be overburdened suddenly. In addition to replacement of blood loss, it is considered safe in infants to give ten milliliters of fluid per pound in one hour, and not more than five milliliters per minute. In cases of shock, other causes of circulatory collapse must be considered, such as cardiac anomalies, intracranial hemorrhage from birth trauma and anoxia from pulmonary conditions. With shock, the use of vasopressors is a last ditch procedure and usually has little effect. Surgery is the only chance of survival in these individuals. Thus, there is no choice other than going on with the operation. In some instances, cases have recovered although in shock for periods up to two hours.[21]

IV. *Surgical procedure:* One group of surgeons[7] recommends an extrapleural approach because it permits drainage to the ex-

terior of a leaking anastomosis. But, with a shorter operating period, the transpleural approach results in a superior anastomosis.

As described by Gross,[5] the operative procedure consists of (a) entering the right thorax through the third or fourth intercostal space, by first making an incision from the tip of the scapular to beneath the breast; (b) holding the lung forward, dividing the azygos vein and dissection of a flap of mediastinal pleura; (c) careful mobilization of the proximal and distal esophageal segments sufficiently to allow approximation, but not to diminish blood supply; (d) severance of the tracheo-esophageal fistula and closure of the tracheal opening; (e) anastomosis of the esophageal segments with a cuff of muscularis of the upper segment drawn down over the lower segment to prevent leakage; (f) replacement of the mediastinal flap leaving small drainage vents superiorly and inferiorly; (g) drainage of the pleural cavity by a catheter led out the sixth intercostal space posteriorly; (h) closure of the chest wall.

In rare cases, when it is necessary to bring up the stomach, only enough is drawn up to make the anastomosis without tension. For the prevention of hemorrhage, the left gastric artery is doubly ligated and divided. The stomach is anchored around the periphery of the diaphragmatic hiatus with interrupted sutures to prevent herniation through the diaphragm.

V. *Postoperative care:* Carefully planned postoperative care is, of course, essential in the management of these cases and will have a profound effect upon the prognosis. Although the individual points in management vary from clinic to clinic, certain basic measures are agreed upon by all.

(a). The infant should be placed immediately in an incubator, preferably of the "isolette" type with humidified oxygen. Atmosphere with excess moisture will lessen irritation of the respiratory passages. Again, one must avoid positive pressure which might further embarrass respiration.

(b). Salivary secretions should be aspirated every 15 minutes. Usually, within a day or two, saliva will be swallowed successfully.

(c). Because the infants are too weak to cough up secretions, a thorough tracteal aspiration should be performed about four hours postoperative. This should be done under direct laryngoscopy. It is advisable to carry out this measure regardless of whether there is any evidence of accumulation of secretions in the

bronchial tree. It should be repeated again every six to eight hours if atelectasis develops or secretions accumulate.

(d). Parenteral therapy should be continued as outlined under preoperative care. Strict limitation of total fluids and particularly the sodium containing solutions will minimize the danger of generalized and pulmonary edema.

(e). Antibiotic and vitamin therapy should be continued.

(f). A nurse should be in constant attendance and should record temperature, pulse, and respirations at frequent intervals. This is very important since there may be sudden attacks of cyanosis and apnea resulting from obstruction of the airway by secretions. Should the onset of such episodes not be recognized promptly, sudden death may occur before corrective measures can be applied.

Since it is virtually impossible to maintain an infant's nutritional status on parenteral feedings alone, and since early oral feedings are not possible because of the insecure anastomosis, some other method of postoperative feeding must be found.

Several groups favor the use of an indwelling rubber nasogastric tube, this tube being left in place at the time of surgery. However, Gross[5] and others feel that such a firm rubber catheter may lead to pressure ulceration at the suture line at the time when this area undergoes early postoperative edema. This could lead to increased incidence of leak at the site of anastomosis with eventual stenosis. Gross believes that a gastrostomy should be performed at the same time as the primary anastomosis. This would only add 15 minutes to 20 minutes to the length of the entire operation and would be a useful procedure having almost no complications. The esophagus would be put at rest offering the best possible chance for healing the site of the anastomosis and yet provide a means for adequate feeding. When, in the rare case, the stomach has been used for anastomosis, no gastrostomy is done for easy regurgitation would contaminate the line of anastomosis.[5] Leven,[7] on the other hand, recommends that the gastrostomy be performed after 24 hours and not at the same time as the other major surgery. Garrison and Mayer[22] feel that it is undesirable to add an additional transperitoneal operative procedure, such as a gastrostomy, to the severely ill newborn. They suggest the use of a "polyethylene" nasogastric tube which does not have the disadvantages of a stiff rubber tube.

Regardless of whether a gastrostomy is performed or a naso-

gastric tube is employed, all feedings for the first 24-36 hours should be by the parenteral route. Continuous Wangensteen suction should be applied to the gastric or nasogastric tube keeping the stomach decompressed and thereby avoiding dangerous vomiting. After the second or third day, ten per cent glucose in distilled water may be offered through either the nasogastric or gastrostomy tube. Between 30 ml. to 60 ml. may be given every four hours, and if well tolerated, small amounts of milk formula may be substituted. In any case, one must be careful to avoid giving so much fluid that the stomach becomes fully dilated. If this occurs, regurgitation up into the esophagus may take place and damage the anastomotic site. Oral feedings may be started on the eight to tenth day. However, one must first check the continuity and patency of the esophagus using a small amount of lipiodol instilled into the esophagus (see discussion below). Small amounts of distilled water, or glucose and water, may be offered at first, and later, milk formula with a gradual increase to full feedings by the end of two to three weeks. Gross[5] suggests that the gastrostomy tube be left in place, although clamped off. If oral feedings are taken well it may be withdrawn in five to six weeks.

Roentgen examination is not only essential for the diagnosis of this condition, but is especially important during the postoperative management period. Pneumothorax may occur as a result of the thoracotomy. For this reason, a bedside film should be taken immediately postoperative in all cases. One should be certain that complete expansion of the lungs has occurred, since this will be important from the standpoint of the infant's breathing as well as sealing of the thoracotomy site by the underlying pleura. Any residual air found in the pleural space should be promptly aspirated. Early postoperative pneumonia or atelectasis may not be evident clinically although be well defined on x-ray. Pleural effusion, a rare complication, may also be demonstrated by roentgen examination. If bilateral pneumonitis fails to disappear, there may be recurrence of the tracheo-esophageal fistula or possibly even an additional and unrecognized tracheo-esophageal communication. As mentioned above, before oral feeding are attempted, the esophagus must be examined with the aid of iodized oil. If there is no leakage at the site of the anastomosis, oral administration of small amounts of fluids is started. This examination should be repeated several days later in order to be sure that the esophagus has remained intact. Only then should feedings be progressively increased in amount. Periodic examination of the esophagus is

also important in determining the degree of constriction which may occur at the site of anastomosis. Later, if dilatation of an esophageal stricture is required, x-ray may be useful in controlling the various dilating procedures and in accurately evaluating their results.[16]

VI. *Postoperative complications:* Generally, in the uncomplicated case, the infant may be discharged from the hospital in about three weeks.[5] However, certain major complications may develop, including pneumonitis, leakage at the anastomotic site, severe postoperative coughing, and stenosis of the esophagus.

a. Pneumonitis is the most common postoperative complication and by far the most frequent cause of postoperative death in these infants.

b. Leakage at the anastomotic site, on the other hand, only occurred in sixteen cases of the one hundred and three reported by Leven.[7] The external esophageal fistula which often develops as a result of this leakage usually heals without difficulty. This is in contrast to re-establishment of the primary tracheo-esophageal fistula which is a very serious complication and occurred four times in Leven's series.

c. A dry unproductive cough may develop and is usually related to the primary pulmonary infection often present when the anomaly is first discovered or it may be due to scarring of the posterior wall of the trachea.[5] Commonly, this cough disappears spontaneously as the infant grows older. However, it may persist and mean that some defect is still present as esophageal stenosis or recurrence of the tracheo-esophageal fistula.

d. Stricture of stenosis of the esophagus is not an infrequent complication, usually occurring within the first five or six months of the postoperative period. Gross[5] reported ninety-seven cases in his series; however, only thirty-four required dilatation. This brings up the very important point that just finding stenosis of the esophagus on x-ray examination without clinical evidence of an inadequate swallowing mechanism cannot by itself be used as an indication for dilatation. Many of these cases will spontaneously improve as normal growth and development proceed and require no active measures.[16]

PROGNOSIS

In a survey of the larger series [5, 7, 21, 22] reported in the liter-

ature, two hundred and forty-seven of five hundred and twelve cases have survived by means of surgical correction of this anomaly. This is an overall survival rate of slightly better than 50 per cent. Since the primary procedure has been used, the survival has increased to 57 per cent [5, 7, 23].

It is held[21] that the mortality should be less than 20 per cent if other lesions incompatible with life are excluded. The mortality that occurs is largely beyond the control of the surgeon and anesthetist, that is, death is due to other malformations, hemorrhage or pneumonia.

Prematurity was found in 22 per cent of cases, that is, sixty-eight of three hundred and one cases.[5, 21] Roughly one-third of these survived, the smallest being two pounds fifteen ounces.[5]

In ninety-three of two hundred and eighty-two cases, or 33 per cent,[5, 21] associated congenital anomalies were found, some of which caused death or were a serious threat to life. Most frequent were congenital heart disease, malformation of the anus or rectum, atresia or stenosis of the small intestine and Meckel's diverticulum. In nine per cent of these cases, a second operation was necessary to deal with the associated anomaly.

Thus, there has been marked improvement in the prognosis of this malformation since 1939, when the first case of congenital esophageal atresia was successfully treated.

SUMMARY

We have attempted to correlate the current thinking on the subject, and the basic embryology related to atresia of the esophagus with tracheo-esophageal fistula has been reviewed.

The diagnosis, management and prognosis of this anomaly have been discussed in detail.

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HICCUPS: PATHOGENESIS AND THERAPY. C. CAMPANA. (Policlinico (sez. prat.), 62:169-175, Feb. 7, 1955). The etiology of intractable hiccups is reviewed and the treatment is discussed. Empirical methods, such as digital compression of the eyeballs to the point of mild discomfort for 30 to 40 seconds, pressure on the carotid sinus or the upper lip, traction of the tongue, sudden fright, and induction of vomiting by pharyngeal irritation, have often proved helpful. If these methods fail, since dilatation and irritation of the stomach are common causative factors, gastric lavage or permanent gastric aspiration should be the next step. Intravenous injection of atropine or bromides as well as the administration of ganglioplegics, mainly Largactil, chlorpromazine [10-(γ-dimethyl-aminopropyl)-2-chlorophenothiazine hydrochloride], to depress the central nervous system is also useful. This ganglioplegic has an efficacious preventive action against hiccups that arise during anesthesia or surgical intervention. Inhalation of carbon dioxide through a long rubber tube is also effective in stopping hiccups because it stimulates the respiratory center in the brain. When all these measures fail to bring relief, the most radical but most effective treatment is the temporary blocking of one or both phrenic nerves or of the third, fourth, fifth cervical roots with injection of atropine by the paravertebral approach. If relief is not obtained, crushing of one or both phrenic nerves becomes necessary. —*J.A.M.A.*

DEPARTMENT OF ABSTRACTS

Conducted by
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JOHNSON, J. R.; FURSTENBERG, N. E.; PATTERSON, R.; SCHOCH, H. K. and DAVEY, W. N.: CORTICOTROPIN and ADRENAL STEROIDS AS ADJUNCTS TO THE TREATMENT OF TUBERCULOUS MENINGITIS. (Annals of Internal Medicine, 46:316, Feb. 1957.)

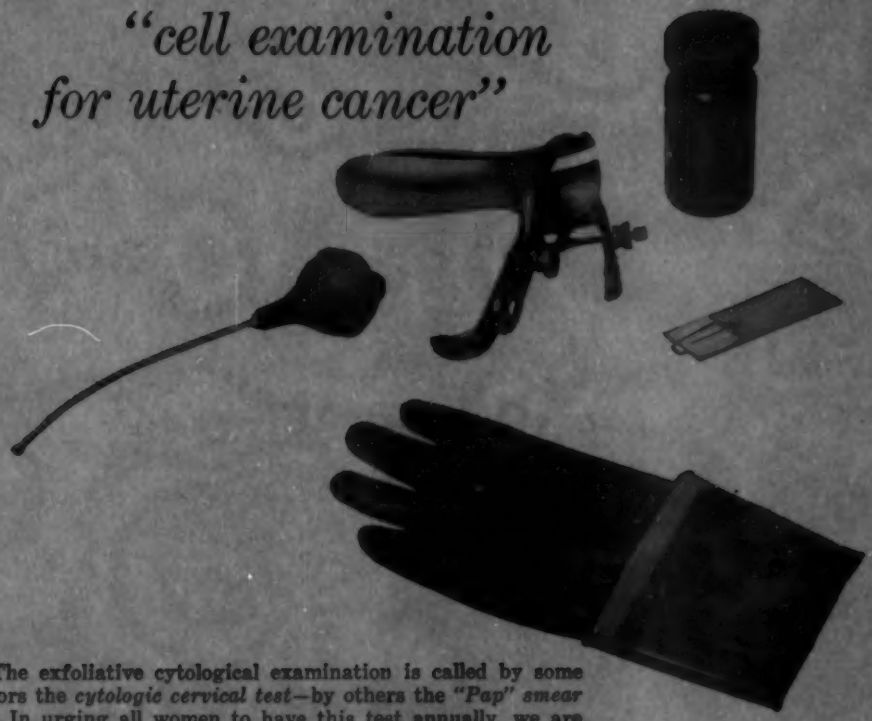
Corticotropin, cortisone and hydrocortisone given in interrupted courses are shown to be correlated directly with marked improvement of symptoms, signs and cerebrospinal fluid findings in three cases of tuberculous meningitis. In a fourth patient, improvement did not occur during one month of routine chemotherapy but became apparent the day after corticotropin was begun. The fifth patient, comatose and in her seventies, had irreversible brain damage, but necropsy revealed complete gross and microscopic healing of her tuberculous infection after two months of cortisone-antimicrobial treatment. Each patient received continuous conventional streptomycin and isoniazid therapy. These five cases are believed to demonstrate the value of combining adrenal hormones with antimicrobial therapy in the treatment of tuberculous meningitis. AUTHORS' SUMMARY.

HOLLAND, W. W. and YOUNG, I. M.: NEONATAL BLOOD PRESSURE IN RELATION TO MATURITY, MODE OF DELIVERY, AND CONDITION AT BIRTH. (British Medical Journal, 5005:1331, Dec. 8, 1956).

Palpation of the brachial artery below a 2.5 cm. inflatable cuff was used to measure the neonatal systolic blood pressure. The normal mean systolic pressure at birth was found to be 69 mm. Hg. rising to 93 mm. at 6 months. Foetal anoxia and abnormal forms of delivery such as caesarean section caused a significant lowering of systolic pressure at birth; the blood pressure of these babies recovered to normal limits within a few days. Drugs given to the mother before delivery and preeclampic toxemia had no effect on the neonatal systolic presure at birth. The systolic pressure of premature babies was lower than that of full term infants and was related to weight. AUTHORS' SUMMARY.

AN EXPERIMENT IN MEDICAL NOMENCLATURE INTRODUCING THE TERM:

"cell examination for uterine cancer"



The exfoliative cytological examination is called by some doctors the *cytologic cervical test*—by others the *"Pap" smear test.* In urging all women to have this test annually, we are calling it the *cell examination for uterine cancer.*

Here are our reasons:

Cytologic cervical test is a term which seems complicated to many women.

"Pap" smear test is simple, but women we have talked to find the word "smear" unpleasant and disturbing, and it may add to their anxieties about pelvic examinations.

Public relations advisors say that broadcasters and editors will dislike "smear"—and TV, radio and the press will be essential to the success of this educational project.

We have considered other terms but have at last agreed on *cell examination for uterine cancer* as the term which simply and accurately describes the keystone of this vitally important program.

This test can help save thousands of women each year. In many parts of the country it is becoming widely accepted as a part of a routine checkup. As fast as county medical societies approve, our local Units will urge women to go to their physicians annually for a *cell examination for uterine cancer.*

AMERICAN CANCER SOCIETY

1 IN 4
NOW
1 IN 3

Ten years ago, only one in four cancer patients was being saved. Today, you, doctor, can expect to save one in three—thanks to your own leadership, a more aware public, improved techniques of diagnosis and treatment. We expect this progress to continue to the point where half of those stricken by cancer will be saved. As yet, science does not have the know-how to save the other half.

That knowledge will come when the riddle of cancer is solved in the research laboratories. To support this vital work, and to carry on its education and service programs, the American Cancer Society seeks $30,000,000 this Spring. We are again appealing to the public to "fight cancer with a checkup and a check."

The check is insurance for tomorrow. The insurance for today is largely in your hands, doctor. Fighting cancer with a checkup is our *immediate* hope for saving lives.

AMERICAN CANCER SOCIETY